KAZIA THERAPEUTICS

Beyond Inhibition.
Reprogramming Cancer Control.

Confidential Investor Presentation

NASDAQ: KZIA | X: @KaziaTx

August 2026

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This presentation contains forward-looking statements, which can generally be identified as such by the use of words such as “may,” “will,” “estimate,” “future,” “forward,” “anticipate,” “plan,” “expect,” “explore,” “potential” or other similar words. Any statement describing Kazia's future plans, strategies, intentions, expectations, objectives, goals or prospects, and other statements that are not historical facts, are also forward-looking statements, including, but not limited to, statements regarding: the timing for interim or final results and data related to Kazia’s clinical and preclinical trials, or third-party trials evaluating Kazia’s product candidates, timing and plans with respect to enrollment of patients in Kazia’s clinical and preclinical programs, the potential benefits of paxalisib, NDL2, MSETC, and EVT801, the potential results of combination studies of paxalisib and other collaborations, the preliminary nature of clinical data from Kazia's ongoing Phase 1b trial of paxalisib in advanced triple-negative breast cancer, including the observed reductions in exhausted T-cells, which are based on a small number of patients and may not be predictive of results in a larger patient population or in later-stage clinical trials, the anticipated timing of topline data from the Phase 1b trial in advanced triple-negative breast cancer and the expected enrollment of patients in such trial, Kazia's plans to expand clinical development of paxalisib into hormone receptor-positive, HER2-negative breast cancer and colorectal cancer, and the timing, cost and results of any such expansion, the potential of paxalisib to act as an immunotherapy sensitizer, remodel the tumor microenvironment, reinvigorate anti-tumor immunity or enable checkpoint inhibitor activity in historically refractory settings, timing for any regulatory submissions or discussions with regulatory agencies, the potential market opportunity for paxalisib, NDL2, MSETC, and EVT801, and Kazia’s strategy and plans with respect to its business and programs. Such statements are based on Kazia’s expectations and projections about future events and future trends affecting its business and are subject to certain risks and uncertainties that could cause actual results to differ materially from those anticipated in the forward-looking statements, including risks and uncertainties: associated with clinical and preclinical trials and product development, the risk that interim and preclinical data may not be reflective of final data, related to regulatory approvals, and related to the impact of global economic conditions. These and other risks and uncertainties are described more fully in Kazia’s Annual Report, filed on Form 20-F with the SEC, and in subsequent filings with the SEC. Kazia undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events, or otherwise, except as required under applicable law. You should not place undue reliance on these forward-looking statements, which apply only as of the date of this presentation.

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Investment Framework

Key elements underpinning our potential for value creation

Early clinical validation creates multiple paths to significant value

Stage IV TNBC clinical observations + potential expansion into >$40B of additional oncology markets

6/6

Stage IV TNBC Patients

Immune Restoration

Reduced and reprogrammed terminally exhausted CD8+ T cells

6/6

Stage IV TNBC Patients

Metastatic Biology Disrupted

Reduced circulating tumor cell (CTC) clusters associated with metastatic dissemination

Extensive Preclinical Data & IP to Support Expanded Indications

HR+ / HER2- breast cancer • Early-stage high-risk TNBC • pMMR CRC

>$40B

Potential Market Opportunity

6/6

Stage IV TNBC Patients with Clinical Benefit

1 Complete Response • 4 Partial Responses • 1 Stable Disease

Ongoing Complete Metabolic Response with Persistent Biological Reprogramming

Reductions in terminally exhausted CD8⁺ T cells and CTC clusters persist alongside complete metabolic response

WHY THIS MATTERS

Early clinical observations support a potentially differentiated mechanism with applicability beyond TNBC.

Not all PI3K/mTOR inhibitors are created equal. Paxalisib's ability to reprogram tumor and immune biology appears distinct and is not a class effect, supporting potential expansion across multiple solid tumors.

Potential Value Creation Pathway

Stage IV TNBC	Additional Data	Broader Indications	Strategic Optionality
Clinical validation	Increased confidence	Expand addressable market	Development / Partnering

Data on file

Kazia Therapeutics Limited

- **Clinical-stage oncology** company focused on developing therapies for **aggressive solid tumors**

- Our technology leverages:
  - Precision pathway inhibition to **reprogram cancer biology, restore anti-tumor immunity**, and **overcome treatment resistance**
  - Next-generation therapeutics that **target multiple layers of cancer and immune control**
  - Multi-modal platforms targeting tumor dormancy designed to **overcome metastases**, **recurrence**, and **resistance** in aggressive and immunotherapy-refractory cancers

Company Highlights

01 Clinical-stage oncology company advancing next-generation differentiated therapies designed to overcome resistance in aggressive and immunotherapy-refractory cancers

02 Lead asset **paxalisib** is an oral, brain-penetrant dual PI3K/mTOR inhibitor for solid tumor indications, including breast cancer, evaluated to date in >550 adult and pediatric patients across Phase 1-3 clinical trials and expanded access programs

03 Advancing **NDL2** – a potentially first-in-class protein degrader designed to target nuclear PD-L1 to overcome immune resistance beyond checkpoint blockade

04 Advancing **MSETC** – a potentially first-in-class SETDB1 inhibitor designed to target SETDB1, a key epigenetic regulator of immune evasion

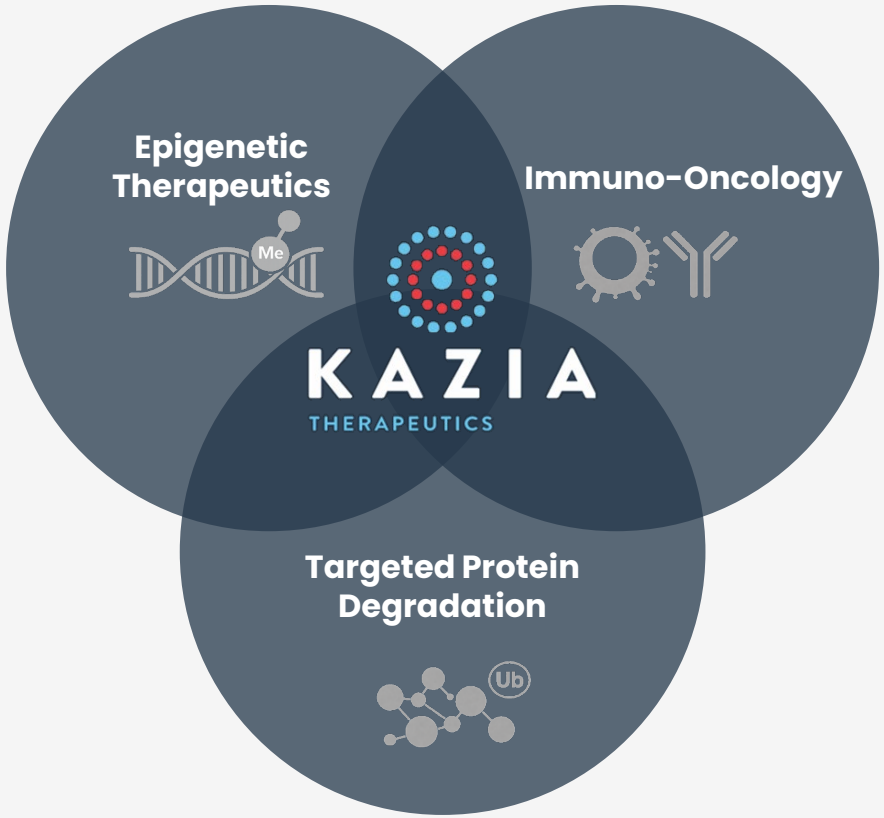
05 Developing **EVT801**, an oral, highly selective VEGFR3 inhibitor targeting tumor lymphangiogenesis and metastatic spread, with strong partnering potential

06
- Cash & Cash Equivalents: Approximately US$46 million (Runway expected to fund planned operations into 2029, excluding additional or expanded trials)
- No outstanding debt
- American Depository Shares Outstanding (Fully Diluted): Approximately 14.1 million

A Differentiated, Multi-Layered Approach

A strategic expansion to a multi-modal platform targeting stable tumor states

Epigenetic Focus & Integration



Paxalisib
Transcriptional Reprogramming

- Alters gene expression programs that drive tumor growth and immune suppression
- Exerts downstream effects on tumor transcriptional programs and immune microenvironment modulation
- Activity extends beyond pathway inhibition, toward tumor-state reprogramming

NDL2
Immune Protein Degradation

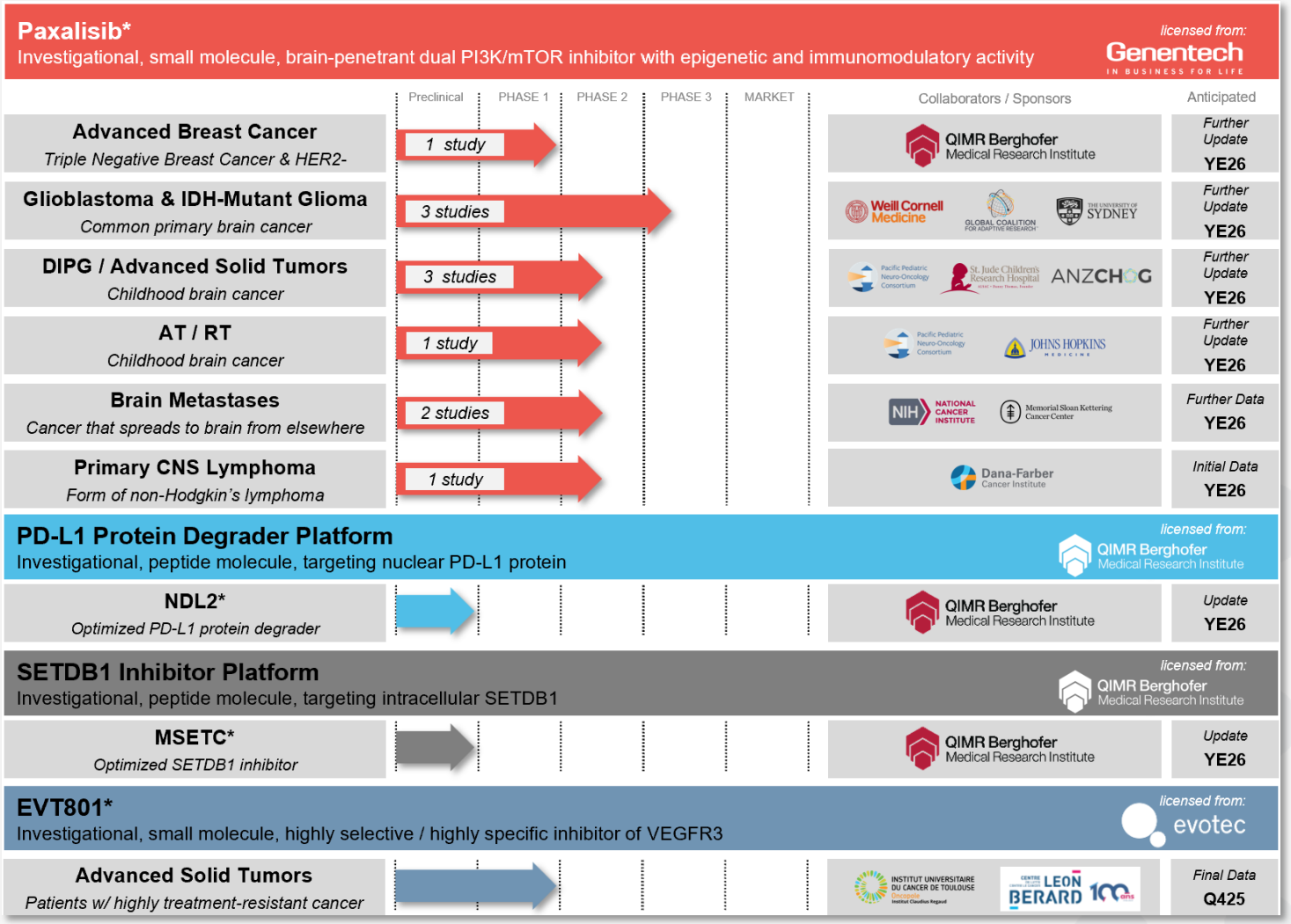
- Eliminates nuclear PD-L1 and overcomes resistance mechanisms beyond antibody-based therapies
- Disrupts immune checkpoint stability and transcriptionally regulated immune resistance pathways
- Intersects with epigenetic immune regulation by targeting nuclear PD-L1 protein (rather than blocking receptor interaction)

MSETC
Chromatin Regulation

- Restores immune visibility by reactivating suppressed signaling pathways
- A chromatin-modifying enzyme implicated in cancer drug resistance, immune evasion, and metastatic progression
- Its integration provides a mechanistic anchor that unifies paxalisib and NDL2 under a coherent tumor-state epigenetic strategy

Pipeline Overview

High-value indications with strong partnering potential



*Product candidates (not approved); IDH: Isocitrate dehydrogenase, DIPG: Diffuse Intrinsic Pontine Glioma, AT / RT: Atypical Teratoid Rhabdoid Tumor, CNS: central nervous system, TNBC: triple negative breast cancer, VEGFR3: vascular endothelial growth factor receptor 3

Overview

Asset Overview	Brain-penetrant, oral, once daily small molecule dual PI3K/mTOR inhibitor with epigenetic and immunomodulatory activity
Origin	Discovered by Genentech (GDC-0084); In-licensed by Kazia Therapeutics
Development Stage	Clinical
Lead Indications	Advanced Breast Cancer (ABC, Phase 1), Glioblastoma (GBM, Phase 3), Childhood Brain Cancer (Phase 2 / Preclinical)
Formulation	Oral, capsule
Key Differentiators	Oral once-per-day dual PI3K/mTOR inhibitor in clinical development for ABC; addresses key mechanisms of immunotherapy resistance; as well as ability to cross the blood brain barrier (BBB) as a potential therapy for primary and secondary brain cancers
Mechanism of Action	PI3K/mTOR kinase inhibitor that disrupts core oncogenic signalling driving tumor growth and survival, while also affecting tumor epigenetic regulation, transcriptional state, and the immune microenvironment beyond its core kinase inhibition
FDA Designations	Fast Track and Orphan Drug for GBM; Fast Track for PI3K-altered brain metastases with radiation; and Rare Pediatric Disease and Orphan Drug for select pediatric gliomas, including diffuse intrinsic pontine glioma (DIPG) and atypical teratoid/rhabdoid tumor (AT/RT)
IP	Patent protection projected until 2043; coverage in over 40 jurisdictions, including U.S., Europe, Japan, China, and India; three patent families: paxalisib molecule *per se* (granted), synthesis of paxalisib (granted), and secondary medical uses / combination treatments (pending)

Expansion Opportunity

Targeting difficult-to-treat cancers where therapeutic options remain limited

Glioblastoma (GBM)

- PI3K/mTOR signaling pathway is a central regulator of cell growth and is hyperactivated in approximately ~85% to 90% of glioblastoma cases.
- Unlike other PI3K inhibitors approved for peripheral cancers, paxalisib crosses the blood-brain barrier overcoming a major obstacle in CNS drug delivery.

Childhood Brain Cancer / Metastasis

- PI3K/mTOR signaling pathway is a central regulator of cell growth and is hyperactivated in approximately ~65% to 70% of pediatric high-grade brain tumors, including DMGs and atypical teratoid / rhabdoid tumors (AT/RT).
- Given the frequent dysregulation of the PI3K/mTOR pathway in pediatric brain tumors and limited effective therapies, paxalisib's ability to cross the blood–brain barrier and combine with multiple treatment modalities presents a compelling therapeutic opportunity.

Advanced Breast Cancer (ABC)

- PI3K/mTOR signaling pathway is frequently dysregulated in breast cancer and associated with tumor proliferation, disease progression, and therapeutic resistance.
- Brain metastases develop in ~25–46% of patients with metastatic TNBC over the disease course, with ~10–15% present at initial metastatic diagnosis.

Expanding Indications

- Broad PI3K/mTOR signaling across solid tumors
- Tumor and tumor microenvironment (TME); emerging data suggests paxalisib can reshape both and potentially overcome resistance and recurrence
- Clear opportunity to expand into HR+ / HER2- breast cancer, early-stage high-risk TNBC, and pMMR CRC

Data on file, market research performed 2021, Glioblastoma Multiforme Treatment Market Size Report, 2034 NCI: Childhood Brain and Other Nervous System Cancer NIH: PI3K Pathway in Pediatric Brain Tumors Strategic Market Research: Pediatric Brain Tumor Market Report Cancer.org 2024 Data Bridge Market Research

Reprogramming Cancer, Not Only Treating It

Targeting the biology of metastasis, resistance, and relapse

Global Unmet Need

- >90% of cancer deaths are driven by metastatic disease
- **HR+ / HER2- Breast Cancer • TNBC • pMMR CRC**
  Three major global cancers with limited effective treatment options
- Current treatment modalities do not address critical drivers of metastasis and relapse

The Opportunity

- **HR+ / HER2- Breast Cancer***
  - Resistance and relapse near universal
- **TNBC***
  - Limited systemic options post I/O
- **pMMR CRC***
  - I/O largely ineffective

* metastatic

Paxalisib Biological Breakthrough

- **CTC Clusters** → Metastatic seeds conventional therapy cannot eliminate
- **Terminally Exhausted T Cells** → Immune escape mechanism SOC cannot reverse

Both are epigenetically hardwired, requiring a fundamentally different approach.

Our Unique Positioning

- Deep IP
- Clinically ready
- Novel liquid biomarkers
- Precision oncology regime

THE SOLUTION

We are targeting the critical seeders of metastasis and reprogramming the immune system early enough to potentially change the trajectory of disease across major solid tumors with global unmet need. We have the biology, the IP, the clinical readiness, and the biomarkers to demonstrate its effect.

Why Current Treatments Fail

The biology always wins…until now

	CHEMOTHERAPY	IMMUNOTHERAPY	RADIOTHERAPY
CTC Clusters	Persist post-treatment	Checkpoint-resistant (Szczerba, Nature 2019)	Paradoxically mobilized (Luo, Cancer Letters 2021)
Immune Exhaustion	Deepens post-chemo (Valpione, Nat Cancer 2020)	Epigenetically fixed (Pauken, Science 2016)	Worsens in the tumor microenvironment (Twyman-Saint Victor, Nature 2015)
Net Effect	✗ **Seeds survive**	✗ **Evasion persists**	✗ **Metastasis accelerates**

Current Biological Reality

- **Drivers of metastasis and relapse:**
  - **CTC clusters** → survive every standard treatment modality
  - **Immune exhaustion** → epigenetically hardwired, not reversed by I/O alone
- **Current therapies** → do not address either

Future Biological Reality

- **Target the epigenetic program** driving both the CTC clusters and immune exhaustion
- **Intervene early enough** to change disease trajectory
- **Deliver durable immune response** – not just a transient response

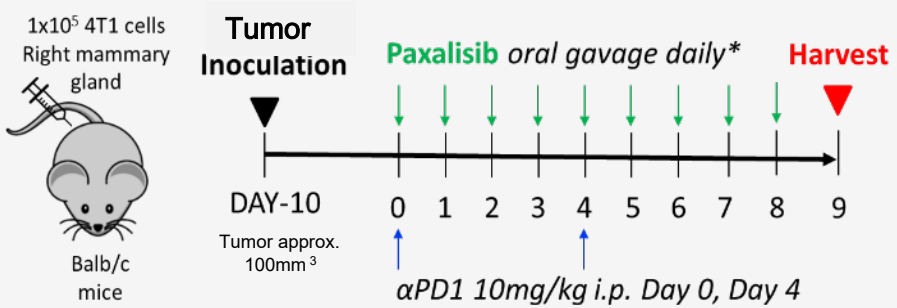
Standard treatments do not address the two critical drivers of metastasis and relapse. Paxalisib is designed to target both.

Consistent & Statistically Significant Signals

Preclinical studies combining paxalisib with either checkpoint inhibitor or PARP inhibitor resulted in highly consistent and statistically significant signals of efficacy.

4T1 Mouse Model

- Standard model for TNBC
- Immunotherapy resistant model
- Highly tumorigenic and invasive



Checkpoint Inhibitor

- Reduced tumor volume
- ↓ Lung metastases
- ↓ Lymph node metastases
- ↓ Liver inflammation
- ↓ Lung inflammation
- ↓ Liver and spleen EMH
- No observed toxicity

PARP Inhibitor

- Reduced tumor volume
- ↓ Lung metastases
- ↓ Lymph node metastases
- ↓ Liver inflammation
- ↓ Lung inflammation
- ↓ Liver and spleen EMH
- No observed toxicity

Data on file, * αPD1: pembrolizumab or "pembro"

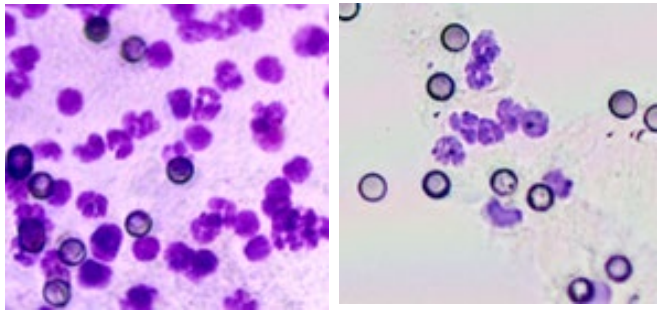
New Data: Preclinical Efficacy in TNBC

Paxalisib targets the biology of relapse and recurrence

Paxalisib + Immunotherapy targets primary metastatic driver → above & beyond direct tumor targeting

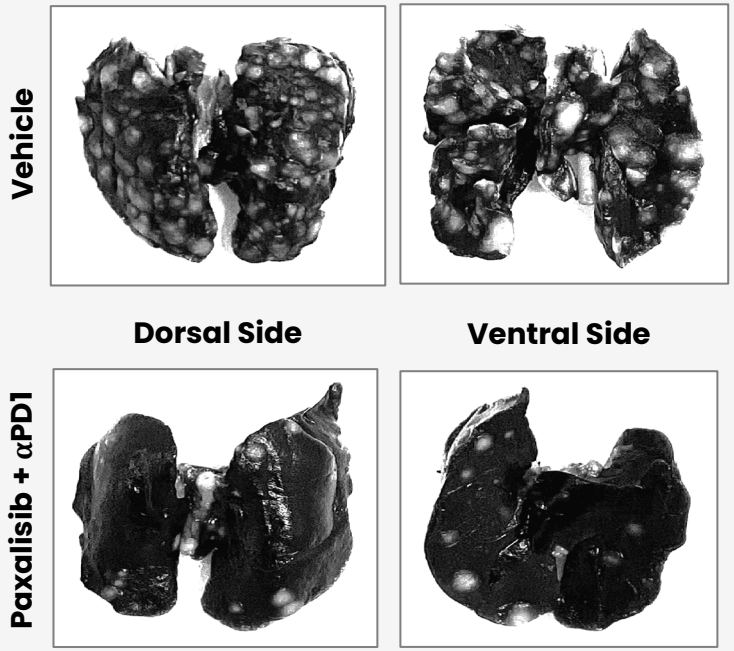
Reduced CTC Clusters

Vehicle control | **Paxalisib + αPD1**



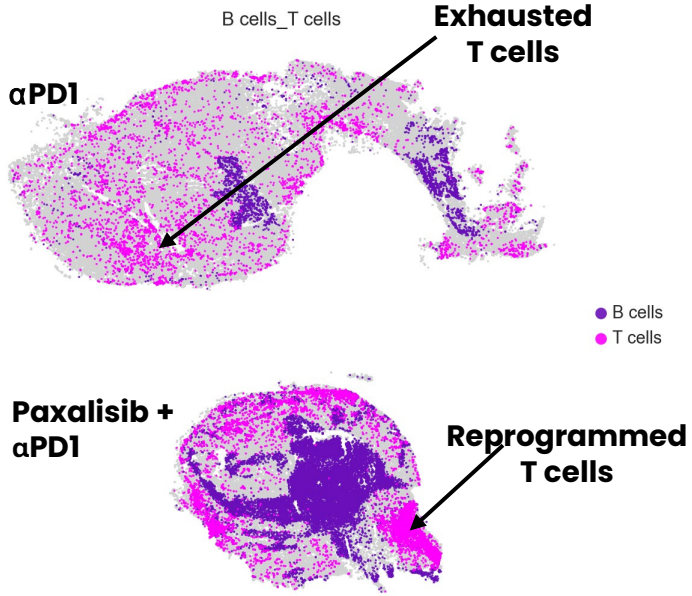
Treatment Group	Animal No#	Circulating Tumor Cells (CTCs) Counts
Vehicle Control	1	>1500
	2	>1500
	3	>1500
Paxalisib + αPD1	1	3
	2	12
	3	295

Reduced Local / Regional / Distant Recurrence



Increased Immune Reprogramming

Spatial protein and ATAC-RNA evidence



Data on file, 4T1 metastasis model

Kazia-Sponsored Clinical Phase 1b Study Overview

Multi-center, open-label, randomized trial

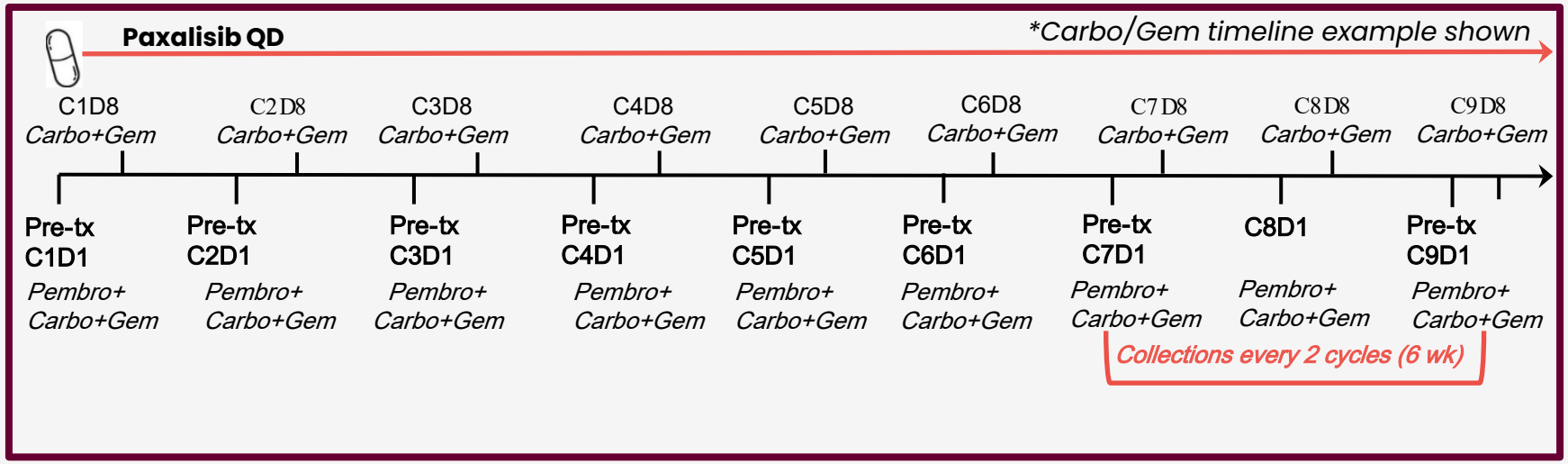
Arm B

- Recurrent, unresectable or metastatic TNBC
- Confirmed that tumors express PD-L1 with a combined positive score (CPS) ≥ 1
- Meet all current prescribing criteria for commencing pembrolizumab therapy

Total enrollment: 36

Paxalisib + Pembrolizumab / Chemotherapy (21-Day Cycle)

- Paxalisib 15mg QD + pembrolizumab 200mg + clinician's choice of IV chemotherapy QW3, n=18
- Paxalisib 30mg QD + pembrolizumab 200mg + clinician's choice of IV chemotherapy QW3, n=18



Endpoints

Primary

- Safety and tolerability of paxalisib in combination with pembro / chemotherapy
- Determine recommended Phase 2 dose (RP2D)

Secondary

- Progression free survival and overall response rates
- To assess the utility of novel liquid biopsy assessments

Trial ID: ACTRN12624001340527

Early Clinical Trial Results

Combination regimen results in >50% reduction in CTCs and early clinical responses

First-in-human data reflects mechanistic synergy consistent with the preclinical data.

Patient One Profile

- 61-year-old female with metastatic TNBC localized to the left upper lobe of the lung

Results at End of Cycle 1

- >50% reduction in single total circulating tumor cells count
- Comparable reduction in CTC clusters (these aggregates are associated with heightened metastatic potential)
- Reduction in the mesenchymal phenotype of the remaining CTCs; this phenotype is one of the hallmarks of aggressive metastatic seeding cancer cells

Preliminary Clinical Responses

- 1 expanded access patient achieved a confirmed complete metabolic response following re-treatment with pembrolizumab / chemotherapy plus paxalisib (30mg)
  - 44-year-old female with metastatic TNBC
  - Previously received pembrolizumab / chemotherapy; experienced disease progression involving bone and lung metastases in early 2025
  - Ineligible for formal trial due to prior pembrolizumab exposure
  - Re-treated under an expanded access protocol beginning in August 2025
- 2/2 evaluable trial patients achieved confirmed partial responses (iRECIST)
- Responses reported in patients with visceral and multi-organ metastases
- Median treatment duration at data cut-off: ~6.1 months

Safety

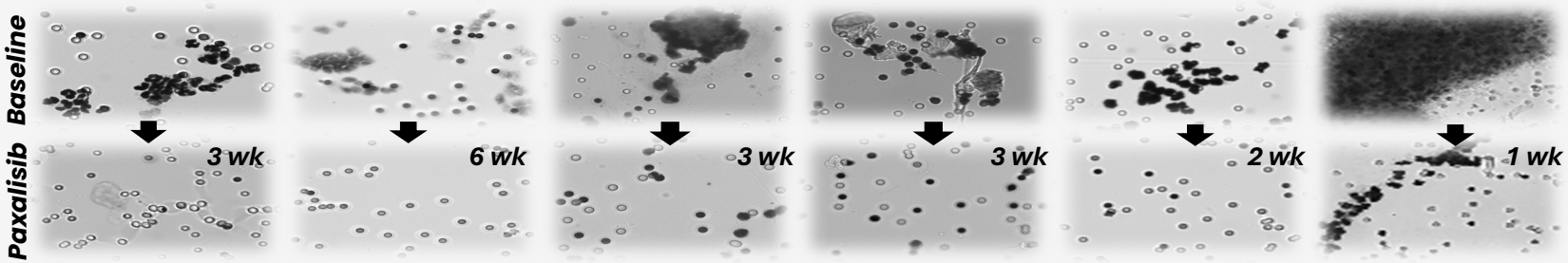
- Generally well tolerated at 30mg daily
- Majority of adverse events assessed as unlikely related to paxalisib
- All paxalisib-related events were mild to moderate to date

Trial ID: ACTRN12624001340527 Kazia Press Release 10 July 2025 Kazia Press Release 27 January 2026 SABCS 2025 poster presentation

New Data: Stage IV TNBC Highlights

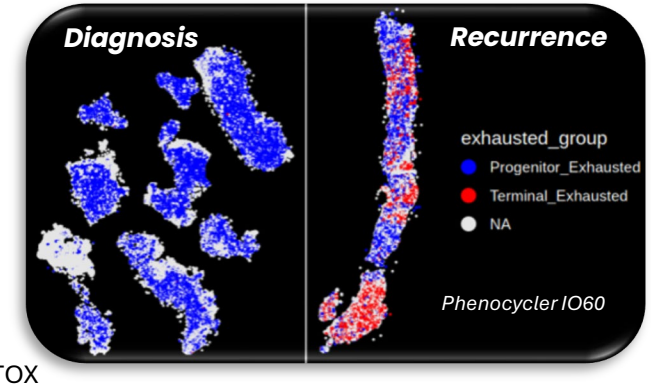
Translational evidence in 6/6 evaluable patients: paxalisib targeting CTC clusters and terminally exhausted T cells

1 Early disruption of CTC clusters by paxalisib combination treatment in Stage IV TNBC



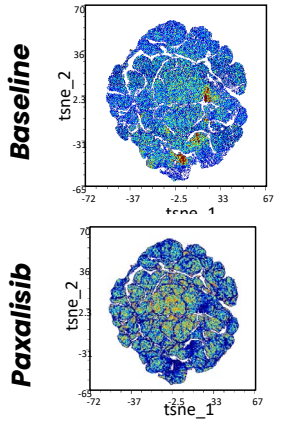
2 Increased terminally exhausted CD8⁺ T cells in progression of TNBC

TNBC disease progression is associated with increased CD8⁺PD1⁺LAG3⁺TOX⁺ terminally exhausted T cells

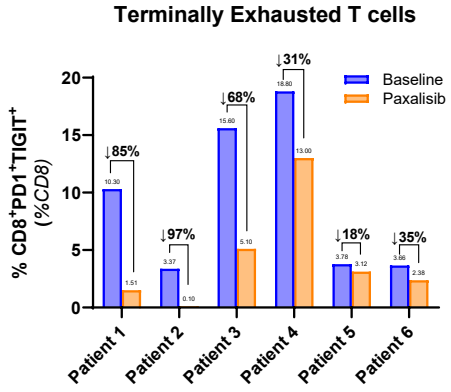


Progenitor: CD8, PD1, TCF-1
Terminal: CD8, PD1, LAG3, TOX

3 Paxalisib rapidly reduces terminally exhausted CD8+ T cells in Stage IV TNBC after 1-3 week treatment



- ↓ PD1
- ↓ TIGIT
- ↓ TIM3
- ↓ LAG3
- ↓ CD160



Data on file

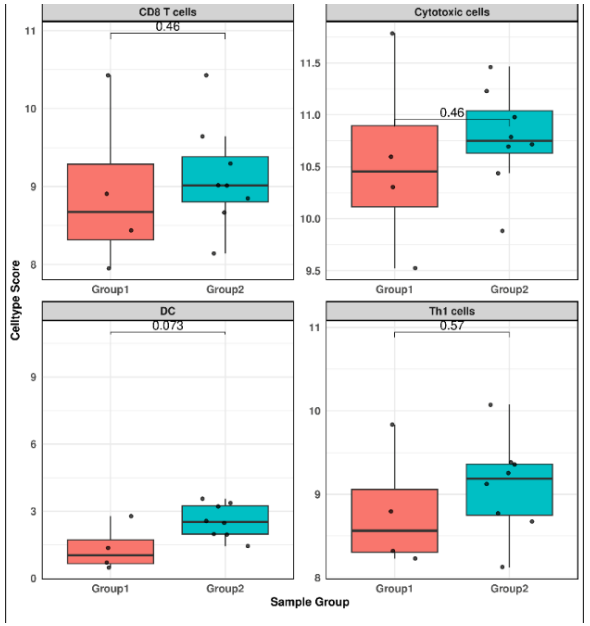
KAZIA THERAPEUTICS

New Data: Stage IV TNBC Highlights (Cont.)

Translational evidence: paxalisib combination reinvigorating the immune landscape

Nanostring RNA profiling of PBMCs isolated from longitudinal patient bloods pre- and post-paxalisib treatment

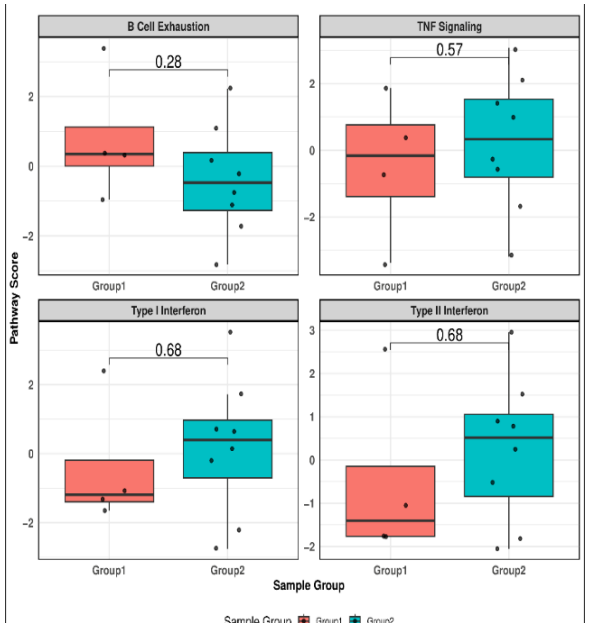
Increased cytotoxicity, IFNγ, DCs



Group 1: Pre-treatment Group 2: Post-treatment

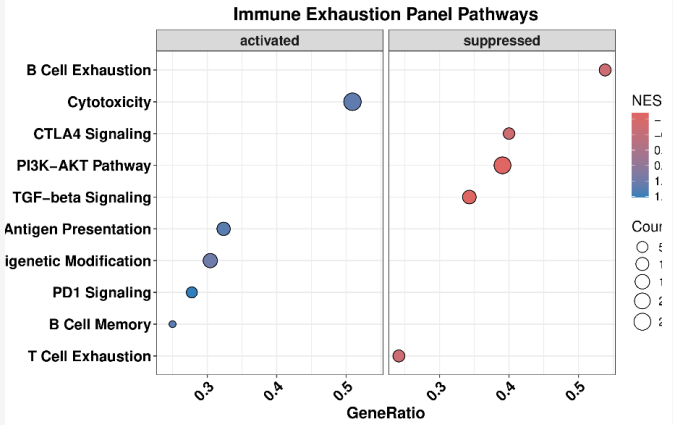
- Increased anti-tumor immune cells
  - ↑ Cytotoxic T cells
  - ↑ Th1 IFNg producing cells
  - ↑ Antigen presenting DCs

Increased antigen presentation, reduced B cell exhaustion



- ↑ Activation of Type I/ II IFN
- ↑ TNF signalling indicative of enhanced cytotoxicity
- ↑ Antigen presentation
- ↓ B cell exhaustion

Inhibition of T cell exhaustion, increased adaptive immune activation



- Global activation of adaptive immune responses
- Inhibition of immunosuppressive signalling (B and T cell exhaustion)
- Confirmation of paxalisib induced inhibition of its target, the PI3K-AKT pathway

Data on file

New Data: Stage IV TNBC Highlights (Cont.)

Early efficacy readout predicts complete metabolic response

MAR 25, 2025

PET Scan

Multiple bilateral pulmonary nodules

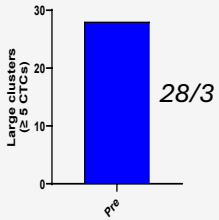
New FDG avid skeletal metastatic disease

JUL 23, 2025

BLOOD

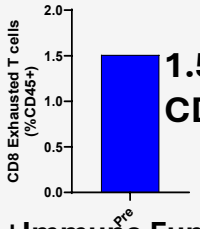
High CTC Clusters

(Large clusters ≥5 CTCs)



28/3 ml

High % Ex T cells



1.5%/ CD45+

↓Immune Function

Low Granzyme A,B

High IL-6 (CTC inducer) (Plasma)

AUG 6, 2025

Paxalisib

+

Pembrolizumab

+

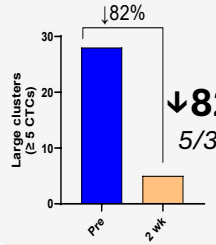
Chemotherapy

Treatment Started

AUG 20, 2025

BLOOD

Reduced Clusters

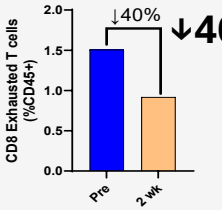


↓82%

5/3 ml

Mesenchymal Vim+NRF2+Snail+ **↓92%**

Reduced Ex T cells



↓40%

↑Immune Function

↑ Granzyme A,B

↓IL-6 (CTC inducer) (Plasma)

AUG 21, 2025

PET Scan

Reduction in pulmonary nodules

Resolution of the FDG activity in the left ilium

No new FDG activity within the axial or appendicular skeleton

NOV 10, 2025

PET Scan

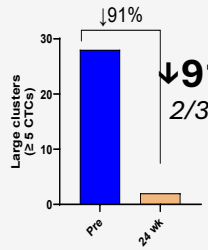
COMPLETE RESPONSE

No evidence of metastatic malignancy

JAN 21, 2026*

BLOOD

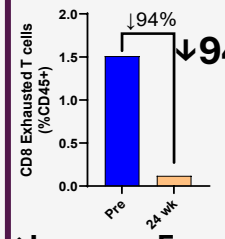
Reduced Clusters



↓91%

2/3 ml

Reduced Ex T cells



↓94%

↑Immune Function

↑ Granzyme A,B

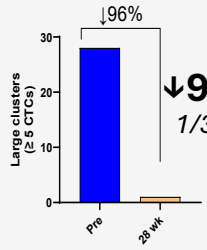
↓IL-6 (CTC inducer) (Plasma)

***PET Scan confirmed CR**

FEB 26, 2026

BLOOD

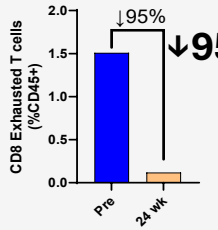
Reduced Clusters



↓96%

1/3 ml

Reduced Ex T cells



↓95%

↑Immune Function

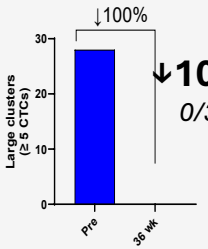
↑ Granzyme A,B

↓IL-6 (CTC inducer) (Plasma)

APR 15, 2026

BLOOD

Reduced Clusters



↓100%

0/3 ml

↑Immune Function

↑ Granzyme A,B

↓IL-6 (CTC inducer) (Plasma)

AUG 4, 2026

PET Scan

COMPLETE RESPONSE

No FDG active locally recurrent, nodal metastatic, or avid distant metastatic disease.

SCAN Matched | **SCAN Predicted** | **SCAN Predicted**

Data on file

KAZIA THERAPEUTICS

New Data: Stage IV TNBC Individual Patient Responses

Patient	Age	Dose	Disease Sites	Response	Time to Response
1	61	30mg	1 target lesion, left upper lobe of lung	Partial	~5 months
2	47	30mg	Lung, liver, bone, lymph node lesions	Partial	~3 months
3	45	15mg	5 target lesions	Partial	~3 months
4	70	15mg	2 target lesions	Stable disease	~5 months
5	43	30mg	CNS target lesions	Partial (Near complete resolution of dural metastases)	~6 months
6	44	30mg	Lung and skeletal target lesions	Complete	~3 months

Key Takeaway:
Responses were observed across visceral, skeletal and CNS metastases.

Data on file

New Data: Stage IV TNBC Safety Profile Supports Continued Clinical Development

0

Paxalisib-Related Serious Adverse Events (SAEs)

0

Suspected Unexpected Serious Adverse Reactions (SUSARs) or Significant Safety Issues (SSIs) Reported

13

Total SAEs Reported

- 1 patient had 1 SAE
- 1 patient had 3 SAEs
- 1 patient had 9 SAEs
- 3 patients had 0 SAEs

Reported SAEs:

Unrelated (10)

Port-related infection (polymicrobial port colonization)
Chest wall pain (non-cardiac)
Parainfluenza
Lung infection
Nausea
Fatigue
Cather-related infection
Diarrhea
Sepsis (2 events)

Unlikely Related (3)

Infectious colitis / diarrhea
Diarrhea
Low-grade fevers (Temps: 37.5° - 37.9° C / 99.5° - 100.22° F)

No SAEs were considered treatment-related and no suspected serious safety signals were reported.

Data on file

New Data: Stage IV TNBC Highlights (Cont.)

Clinical evidence: first-in-human signals from six evaluable patients

100% of evaluable patients (6/6) showed clinical benefit.

Selective immune reinvigoration

- Terminally exhausted $CD8^+$ T cells reduced in **100% of evaluable patients (6/6)**
- **Median 51% reduction** (range 18–97%) within ~3 weeks
- **Total $CD8^+$ T-cell numbers unchanged**, indicating selective reduction of dysfunctional T cells

Robust disruption of metastatic CTC clusters

- **100% of evaluable patients (6/6)** showed reductions in CTC clusters
- **Median 83% reduction** (range 76–90%) by 6–7 weeks of treatment

Clinical implication

- Early, consistent pharmacodynamic responses suggest **paxalisib extends beyond PI3K/mTOR inhibition**
- Supports our hypothesis of paxalisib's ability to target **immune dysfunction** and **metastatic dissemination**, two key drivers of TNBC
- One expanded access patient demonstrated durable complete metabolic response and robust reductions in CTC clusters and exhausted terminally exhausted $CD8^+$ T cells

Data on file

Advancing Paxalisib Programs

One biological opportunity, multiple high-value markets

HR+ / HER2- BREAST CANCER Large population • Relapse • CNS disease	TNBC Metastasis • Brain metastases • Immune escape • Recurrence	pMMR CRC Large population • Limited I/O response • Resistance • Major unmet need
• Large, biologically defined population with substantial unmet need in advanced / metastatic disease • Represents ~60-70% of all breast cancers • ~322,000 new invasive cases of breast cancer are expected in the US alone in 2026 • 5-year relative survival rate of ~35% for distant HR+ / HER2- disease • Predicted market of ~$17B by 2030	• Rare, fast-growing, and aggressive form of breast cancer • Accounts for approximately 10-15% of all breast cancer • 5-year survival rate of 12% for metastatic TNBC • Predicted market of ~$6B by 2030	• ~85% of colorectal cancers is pMMR / MSS, representing the overwhelming majority of CRC patients • ~159,000 new cases are expected in the US alone in 2026 • 5-year relative survival rate of 13-18% for distant stage CRC • Predicted market of ~$18B by 2030

Innovation Highlights:

- IP filed on all three indications
- Compelling preclinical data
- Ready for clinical advancement
  - Trial design with comparative drugs
  - Liquid biopsy early efficacy readout biomarkers
- PI clinicians in place (multi-center)
- Rapid recruitment (12 months)

NIH, American Cancer Society, Cancer.gov, published literature/DelveInsight

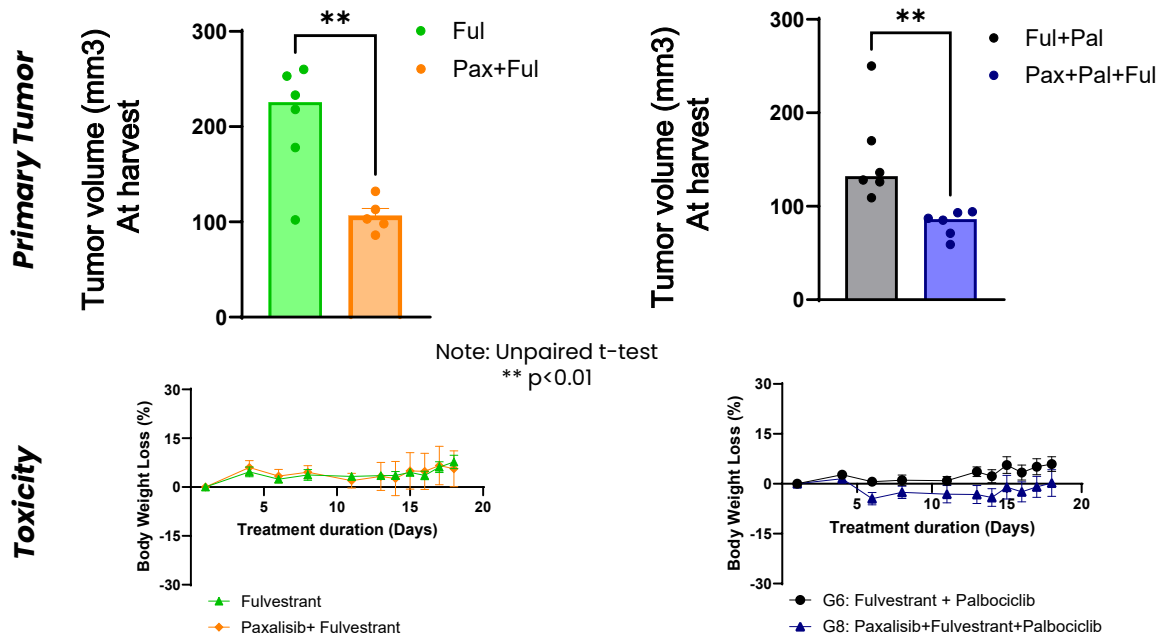
New Data: HR+ / HER2- Innovation & Highlights

Key Highlights

- Patent protected
- Extensive preclinical safety and efficacy evidence supporting paxalisib in combination with standard of care (SOC) in HR+ / HER2- breast cancer patients (both wild-type and mutant)
- Subpopulation with elevated novel PI3K/mTOR marker enriched in HR+ / HER2- breast cancer that is tightly coupled with poor survival identified; axis uniquely targeted by paxalisib
  - Identified a novel epigenetic MOA that allows paxalisib (and not gedatolisib) to uniquely sensitize the CDK4/6 resistant subpopulation to overcome recurrence and metastases
  - Abstract submitted to upcoming conference
- Potential for paxalisib to treat HR+ / HER2- patients with brain mets accounts for 10% HR+ / HER2-
- Clinical-trial ready

Scientific Evidence

Paxalisib + Fulvestrant +/- CDK4/6 inhibitor reduces tumor burden without toxicity

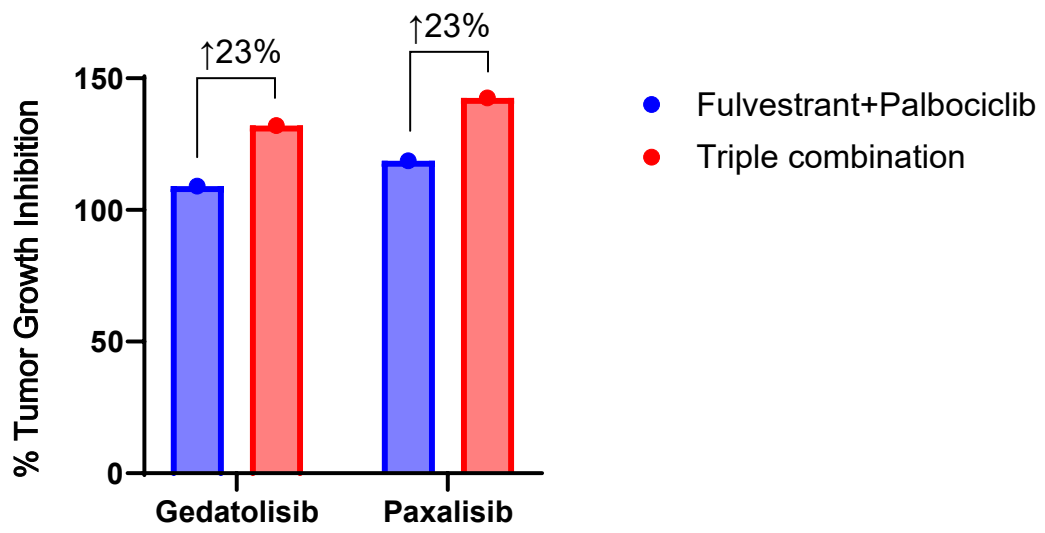


Data on file

New Data: HR+ / HER2- Innovation & Highlights (Cont.)

Scientific Evidence

Comparison of tumor growth inhibition following treatment with Gedatolisib and Paxalisib in combination with Fulvestrant + Palbociclib in the MCF7 HR$^+$ xenograft model



Paxalisib shows identical efficacy to gedatolisib in preclinical studies.

Data on file

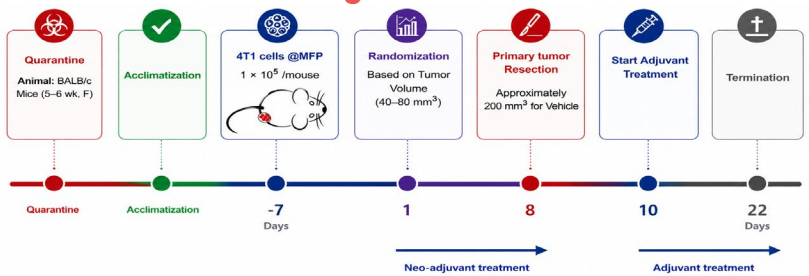
New Data: Stage IIb/III TNBC Innovation & Highlights

Key Highlights

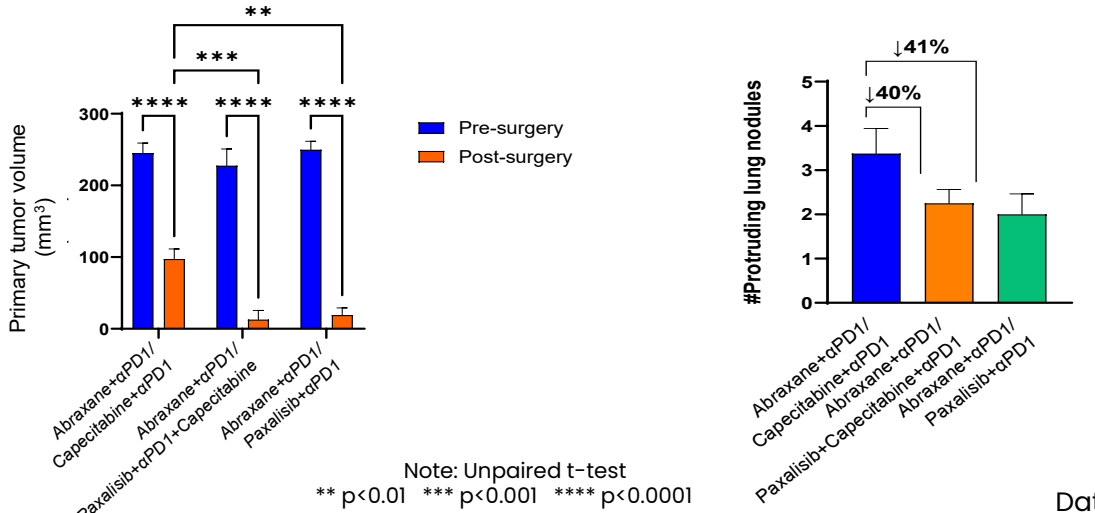
- Patent protected
- Significant residual disease and local recurrence suppressed by paxalisib combination therapy in the 4T1 neoadjuvant / adjuvant surgical metastasis model
- Significant reduction in distant metastatic recurrence
- A quarter-dose chemotherapy combination cuts tumor burden without toxicity
- Patient CTC clusters disrupted ex vivo, with attenuation of the aggressive phenotype in early-stage TNBC
- Novel liquid biomarkers to select patients and read out response early
- Early access partners for spatial ATAC sequencing
- Excitingly, epi-reprogramming of durable immune responses seen for the first time in the lymph node
- Clinical-trial ready — protocols in place

Scientific Evidence

Establishment of a clinically relevant preclinical neoadjuvant / adjuvant 4T1 surgical model



Paxalisib combination therapy suppresses residual disease, local and distant recurrence in a preclinical neoadjuvant / adjuvant 4T1 surgical model



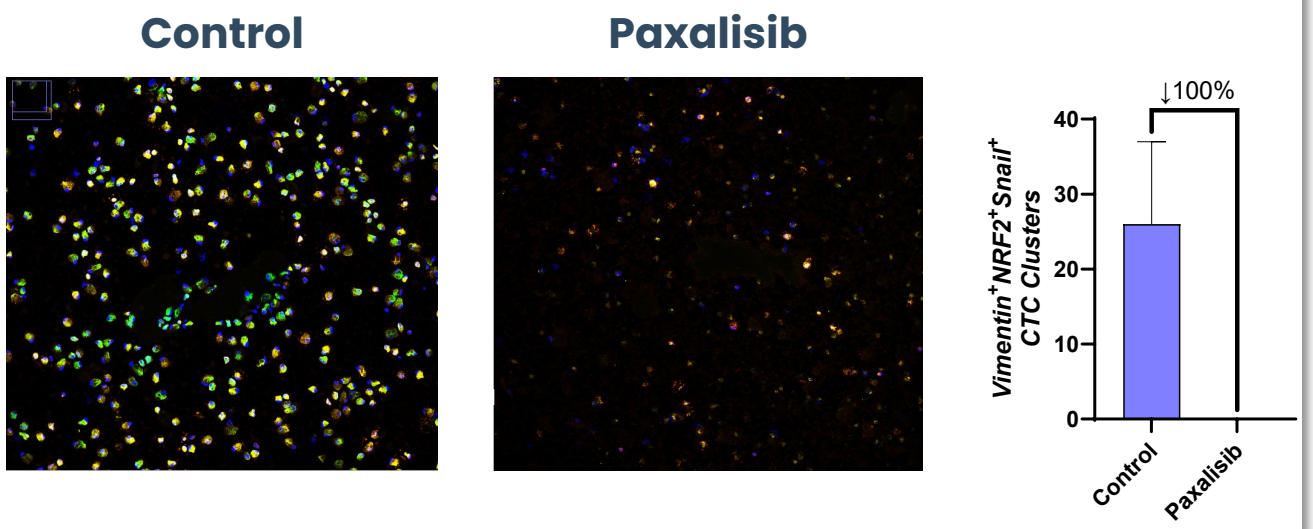
Note: Unpaired t-test
** p<0.01 *** p<0.001 **** p<0.0001

Data on file

New Data: Stage IIb/III TNBC Innovation & Highlights (Cont.)

Scientific Evidence

Ex-vivo Disruption of Circulating Tumor Cell (CTC) Clusters and Attenuation of Aggressive Phenotype Following Paxalisib Treatment in Early-Stage TNBC



Paxalisib disrupts patient-derived CTC clusters ex vivo — the same effect seen in the pre-clinical model.

Data on file

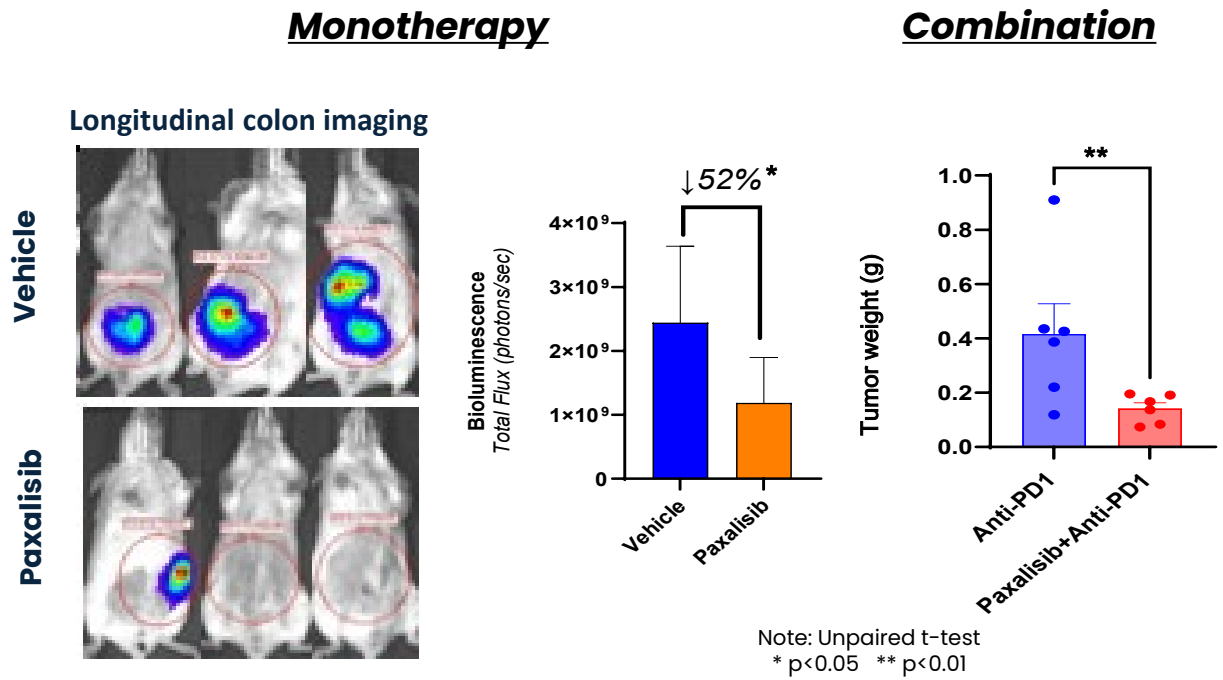
New Data: pMMR CRC Innovation & Highlights

Key Highlights

- Patent protected
- 52% reduction in tumor burden with paxalisib as a single agent (p = 0.035), well tolerated throughout
- A further 50% reduction in tumor volume when added to anti-PD-1, versus anti-PD-1 alone (p = 0.022)
- Novel liquid biomarkers to select patients and monitor response, where none exist today
- Targets the pMMR majority — the 85-90% of colorectal cancer patients who derive limited benefit from checkpoint inhibitors today
- Early access partners for spatial ATAC sequencing
- Clinical-trial ready — protocols in place

Scientific Evidence

Paxalisib monotherapy and αPD1 combination suppresses primary tumor burden in pMMR+ CRC preclinical models



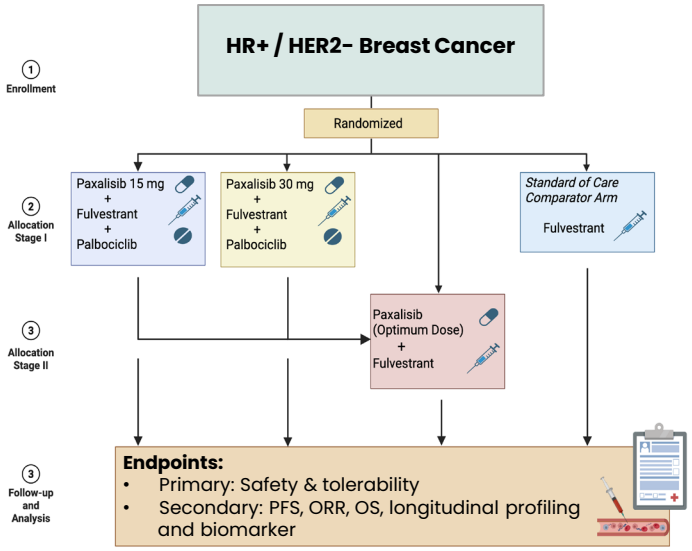
Note: Unpaired t-test
* p<0.05 ** p<0.01

Data on file

Clinical Trial Ready

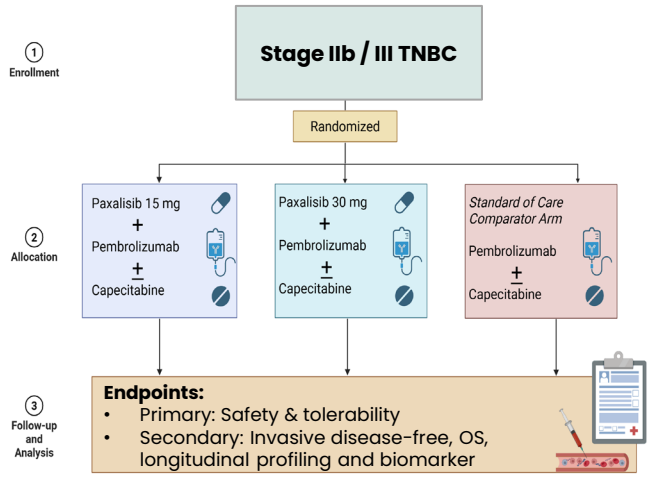
HR+ / HER2- Breast Cancer

Paxalisib + Fulvestrant + Palbociclib for pre-treated HR+ / HER2- metastatic breast cancer (Second line setting)



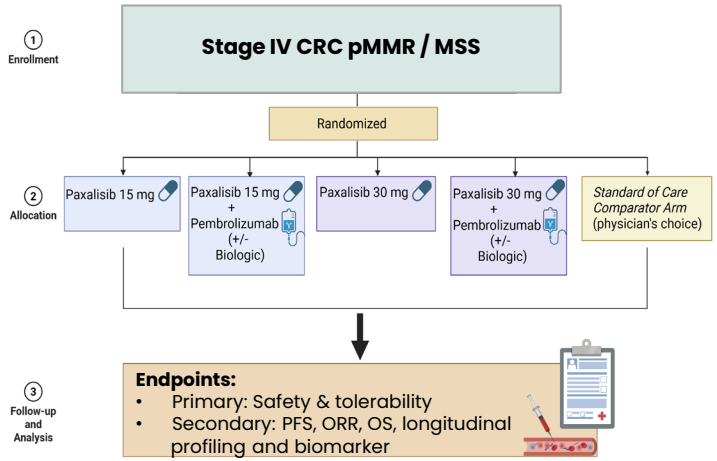
Stage IIb / III TNBC
(in adjuvant, followed by neoadjuvant setting)

Paxalisib + Pembrolizumab + Capecitabine for high-risk Stage IIb / III TNBC in the adjuvant setting (First line setting)



pMMR CRC

Paxalisib Monotherapy and Paxalisib + Pembrolizumab vs. Standard of Care in pre-treated pMMR / MSS metastatic CRC (Third line setting)



Summary & Next Steps

Summary

- Brain-penetrant dual pan-PI3K/mTOR inhibitor in development (Only 2% of small-molecule drugs are brain-penetrant)
- Designed to be best-in-class pan-PI3K pathway inhibitor with modest mTOR activity (dual targeting required to inhibit cancer cell proliferation & migration in TNBC cell model)
- Overcoming metastasis and drug resistance in combination with immunotherapy (epigenetic reprogramming of dormant cancer cells and improved cancer immune visibility)
- Generally well tolerated (evaluated in 550 adult and pediatric patients across Phase 1-3 clinical trials and expanded access programs)

Clinical and Translational Epigenetic Data Demonstrate That Paxalisib:

- Reduces CTC and CTC-clusters as well as aggressive mesenchymal phenotypes (observable impact on metastatic biology)
- Enhances activity when combined with immune checkpoint inhibition (combination-enabling potential)
- Modulates immune exhaustion markers (evidence of tumor microenvironment reprogramming)

On Strategy

- Positioned as a backbone therapy within the emerging category of functional epigenetic reprogramming therapeutics

Next Steps

- **Advanced Breast Cancer**: 1) Provide additional preclinical data and updates throughout the year, 2) ongoing updates from Phase 1b advanced breast cancer clinical study (ACTRN12624001340527) throughout 2026, 3) expand into other clinical indications including HR+ / HER2- breast cancer and early-stage high-risk TNBC and 4) participate in upcoming scientific conferences
- **Colorectal Cancer:** 1) Execute clinical development plan with Phase 2 clinical trial in pMMR CRC to evaluate paxalisib as monotherapy and in combination with pembrolizumab and 2) participate in upcoming scientific conferences
- **Glioblastoma**: 1) Anticipated follow-up FDA Type C meeting to discuss commercial and development path forward and 2) finalize clinical development plan based on FDA feedback
- **Pediatric & Brain Metastasis Programs**: 1) PNOC022 team to complete PK / biomarker data analysis and provide update, 2) data release / updates from other brain met trials, and 3) initiate enrollment for PNOC035 (AT/RT clinical study)

Platform Overview

Complementing paxalisib's transcriptional and epigenetic effects, Kazia's NDL2 program addresses immune resistance at the post-translational regulatory level

Asset Overview	Lead candidate, NDL2, is an optimized bicyclic peptide PD-L1 degrader designed to target nuclear PD-L1, thereby restoring anti-tumor immune responses even in cases of immune evasion or checkpoint inhibitor resistance
Origin	Developed by QIMR Berghofer
Development Stage	Preclinical; IND expected in 18 months
Lead Indications	Breast cancer and non-small cell lung cancer (NSCLC) in early stage and metastatic settings
Formulation	Intravenous infusion
Key Differentiators	First in Class: As the science surrounding PD-L1 degraders is novel (<5yrs), there are no PD-L1 degraders in clinical studies at this time
Mechanism of Action	Target nuclear PD-L1 protein pools Recruits cellular degradation machinery Targets nuclear PD-L1 protein species that cannot be targeted by standard checkpoint inhibitors
FDA Designations	N / A
IP	Initial composition of matter IP filed in 2021

Summary & Next Steps

Innovation Breakthrough

- Selectively targets cancer nuclear enriched PD-L1 protein
- Amongst the first therapeutics to address metastasis-seeding cancer cells resistant to traditional immunotherapy
- Precisely degrades the resistance-associated form of PD-L1, preserving PD-L1 essential for normal immune regulation

Recent Preclinical and Translational Data Demonstrate That NDL2:

- Reduces tumor growth as monotherapy
- Enhances the activity of anti-PD-1 therapy
- Targets resistance mechanisms enriched in immunotherapy-refractory disease
- Preserves normal immune checkpoint function at the cell surface and therefore avoids toxicity associated with anti-PD-L1 agents

On Strategy

- NDL2 is reshaping cancer cell behavior by altering the functional protein landscape rather than simply blocking extracellular signaling
- NDL2 represents a complementary modality to epigenetic and transcriptional reprogramming, extending Kazia's strategy into targeted protein degradation

Next Steps

- Initiate IND-enabling studies
- Integrate a clinical liquid biopsy to enable patient stratification, real-time monitoring, and personalized treatment optimization
- Launch PD-L1 degrader and NDL2 awareness campaign through abstracts, manuscripts, videos, and medical congress presentations

Platform Overview

Complementing paxalisib's transcriptional modulation and NDL2's protein degradation

Asset Overview	Lead candidate, MSETC is an optimized, bicyclic peptide designed to target SETDB1, a key epigenetic regulator of immune evasion. By inhibiting SETDB1, MSETC could restore anti-tumor immune responses, including in tumors resistant to checkpoint inhibitors.
Origin	Developed by QIMR Berghofer
Development Stage	Preclinical; IND expected in 18 months
Lead Indications	Solid tumors with low immunogenicity ("immune cold", e.g. TNBC, NSCLC, CRC)
Formulation	Intravenous infusion
Key Differentiators	First-in-class SETDB1 inhibitor (chromatin-level immune regulator); targets immune evasion; potential to convert "immune cold" tumors into responsive tumors; combination-ready with immunotherapy and targeted agents; intracellular / nuclear mechanism not addressed by antibody therapies
Mechanism of Action	SETDB1 is a histone methyltransferase implicated in immune resistance and immune evasion that silences genes involved in immune recognition and interferon signaling through chromatin modification; designed to suppress immune signaling pathways, increase tumor visibility to the immune system, and potentially reverse resistance to checkpoint inhibitors
FDA Designations	N / A
IP	Initial composition of matter IP filed in 2022

Summary & Next Steps

Innovation Breakthrough

- Targets SETDB1, a chromatin-level regulator that suppresses tumor immune recognition
- Addresses immune evasion at its earliest control layer
- Represents a novel approach to restoring immune signaling in tumors resistant to current therapies

Emerging Preclinical Rationale Supports SETDB1 Targeting

- Reactivates interferon and antigen presentation pathways
- Increases tumor visibility to the immune system
- Sensitizes tumors to checkpoint inhibition in resistant settings
- Targets mechanisms not addressed by current immunotherapies

On Strategy

- Extends Kazia's cancer reprogramming approach to chromatin-level control of tumor behavior
- Complements paxalisib's transcriptional modulation and NDL2's protein degradation
- Enables a multi-layered strategy addressing immune evasion across distinct biological levels

Next Steps

- Advance IND-enabling studies and candidate characterization
- Build awareness and value through continued generation and dissemination of preclinical and translational data supporting the SETDB1 platform and MSETC
- Define optimal combination strategies with checkpoint inhibitors and targeted therapies
- Generate translational data to support patient selection and biomarker-driven development
- Evaluate early partnering opportunities aligned with development milestones

Corporate Highlights

Near-Term Milestones & Anticipated Catalysts

Q3 2026	Q4 2026	Q1 2027	Q2 2027	2027
Readouts from neuro-oncology focused clinical trials in patients with GBM and brain metastases	Update of paxalisib preclinical development into additional breast cancer subtypes	Launch expansion of ongoing clinical trial into other breast cancer populations (HR+ / HER2-, early-stage high-risk TNBC)	Launch expansion into pMMR CRC as monotherapy and in combination with pembrolizumab	Initiate Phase 1 trials for NDL2 and MSETC
Publication / Presentation of translational data supporting therapeutic reprogramming strategy for paxalisib	Anticipated update from FDA discussion regarding paxalisib commercial and development path forward	Clinical efficacy and biomarker data from ongoing Phase 1b breast cancer study	PD-L1 degrader and SETDB1 inhibitor awareness, NDL2 and MSETC IND-enabling studies	

Milestone **Anticipated Catalyst**

Financial Overview

Cash & Cash Equivalents

- Approximately US$46 million at 12/31/25 (Runway expected to fund planned operations into 2029, excluding additional or expanded trials)

No outstanding debt

American Depository Shares Outstanding (Fully Diluted)

- Approximately 14.1 million

Use of Proceeds

- Accelerate ongoing TNBC trial (adding new Australian sites, adding new country / countries)
- Expand ongoing trial: HR+ / HER2- breast cancer, early-stage high-risk TNBC, and CRC
- R&D expense to support ongoing trial expansion (preclinical, ex-vivo) for paxalisib
- Accelerate PD-L1 protein degrader and SETDB1 inhibitor lead assets into IND-enabling and clinical trial
- R&D general expense to evaluate novel targets and expand novel biomarker work
- G&A

Investment Framework Recap

Key elements underpinning our potential for value creation

Early clinical validation creates multiple paths to significant value

Stage IV TNBC clinical observations + potential expansion into >$40B of additional oncology markets

6/6

Stage IV TNBC Patients

Immune Restoration

Reduced and reprogrammed terminally exhausted CD8+ T cells

6/6

Stage IV TNBC Patients

Metastatic Biology Disrupted

Reduced circulating tumor cell (CTC) clusters associated with metastatic dissemination

Extensive Preclinical Data & IP to Support Expanded Indications

HR+ / HER2- breast cancer • Early-stage high-risk TNBC • pMMR CRC

>$40B

Potential Market Opportunity

6/6

Stage IV TNBC Patients with Clinical Benefit

1 Complete Response • 4 Partial Responses • 1 Stable Disease

Ongoing Complete Metabolic Response with Persistent Biological Reprogramming

Reductions in terminally exhausted CD8⁺ T cells and CTC clusters persist alongside complete metabolic response

WHY THIS MATTERS

Early clinical observations support a potentially differentiated mechanism with applicability beyond TNBC.

Not all PI3K/mTOR inhibitors are created equal. Paxalisib's ability to reprogram tumor and immune biology appears distinct and is not a class effect, supporting potential expansion across multiple solid tumors.

Potential Value Creation Pathway

Stage IV TNBC	Additional Data	Broader Indications	Strategic Optionality
Clinical validation	Increased confidence	Expand addressable market	Development / Partnering

Data on file

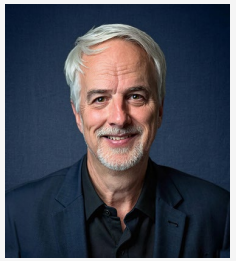
Leadership

Management

Dr. John Friend
Chief Executive Officer
CELLECTAR BIOSCIENCES
HELSINN
ABBOTT

Dr. Sudha Rao
Chief Scientific Officer
QIMR Berghofer Medical Research Institute
sanofi RHONE-POULENC

James Levine
Chief Financial Officer
Cardiff Oncology
CIDARA THERAPEUTICS
Goldman Sachs



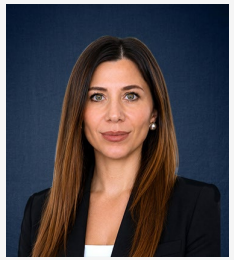
Jeffrey J. Kraws
Head of Corporate Strategy & Development
Bristol Myers Squibb
ABB
ALEX. BROWN

Elissa Hansen
Corporate Secretary
Governance Institute of Australia
Australian Institute of Company Directors

Board of Directors

Robert Apple
Chairman
antares

Steven Coffey
Non-Executive Director
WHK CHARTERED ACCOUNTANTS



Ebru Davidson
Non-Executive Director
QBiotics Group
Thomson Geer Lawyers

KAZIA
THERAPEUTICS

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